Is borderline personality disorder a mood disorder?

Gordon Parker

Summary
Borderline personality disorder is by its very naming positioned as an Axis II personality disorder and thus seemingly distinct from an Axis I mood state. Clinical differentiation of those with a borderline condition and those with a bipolar disorder is commonly held to be difficult, so raising the question as to whether they may be independent or interdependent conditions, and allowing several possible answers.

Declaration of interest
None.

Background

Tyrer\(^1\) has argued that ‘borderline personality disorder is incorrectly classified as a personality disorder’ and would be ‘better placed with the mood disorders’; their clinical differentiation being commonly viewed as difficult is likely to have contributed to this proposition. A key plank to Tyrer’s argument was that only two criteria for borderline personality disorder (BPD, i.e. a pattern of unstable and intense personal relationships, persistent impulsivity) are in any way trait-based. Tyrer also suggested that BPD was an unsatisfactory diagnosis, lacking clinical meaning and occupying a buffer (borderline) zone. Although BPD is positioned in DSM-5 as a personality disorder,\(^2\) Tyrer’s proposition risks BPD moving to a ‘nonaxial documentation of diagnosis’ (p. 16).\(^2\) In personality disorders, I anticipate angst in adopting DSM-5’s and II as respectively capturing differential symptom states and transient paranoid ideation.

Call me old fashioned or call me a splitter (nosologically in this instance) but habituated by DSM-III\(^3\)’s Axes I and II as respectively capturing differential symptom states and personality disorders, I anticipate angst in adopting DSM-5’s move to a ‘nonaxial documentation of diagnosis’ (p.16).\(^2\) In combining Axes I, II and III without the explication of a higher-order conceptual model, disorders and nosological debates are likely to spin increasingly around the lack of axes. As mood disorders are heterogeneous, any consideration as to whether BPD better fits as a mood disorder requires separate consideration of bipolar and unipolar mood disorders.

Is BPD a bipolar mood condition?

One of the most common clinical dilemmas\(^4\) is distinguishing between BPD and bipolar II disorder – where mood swings may be frequent, brief and even reactive. The dilemma is increased if assessment focuses on shared features such as ‘emotional dysregulation’ or mood instability – but reduced by considering longitudinal as well as broader cross-sectional features.

Differential diagnostic pointers\(^5\) include a weighting to ‘breeding true’ – with a family history of bipolar disorder, depression and suicide overrepresented in those with a bipolar condition, and emotional dysregulation states overrepresented in probands of individuals with BPD. Bipolar disorder can (rarely) present in childhood, the modal onset is late adolescence or early adulthood, and generally marked by a sharp ‘trend break’ (from absence of episodes to their overt presence). By contrast, those with BPD tend to show evidence of emotional dysregulation (for example anger, rage, impulsivity) from early years, and which evolves rather than showing a distinct ‘onset’.

Phenomenological distinction is generally informative too. Whereas anger or irritability may occur in mania and hypomania in bipolar disorder, elevated bipolar mood states are more generally characterised by euphoria, grandiosity and feeling creative and bullet proof. Of central importance, anxiety and worry generally disappear or are attenuated as the individual feels ‘care free’. By contrast (and by definition), those with BPD manifest painful extremes of ‘normally’ regulated emotions (principally anger, hostility and anxiety). The utility of weighting differential changes in anxiety is supported by Livesley’s observation\(^6\) that borderline traits are organised around the threat of anxiety, with dysregulation of the threat management system leading to pervasive fearfulness and exaggerated emotions of anger and irritability.

Another point of difference is that, following ‘highs’, individuals with bipolar disorder often experience guilt about indiscretions, which they ‘own’ with some shame. By contrast, individuals with BPD rarely feel guilt or shame following ‘acting out’ episodes, usually positioning them as the deserved and painful consequences of others’ actions – and thus tend to blame others rather than accept any ownership.

Although distinction commonly focuses on the ‘highs’, shared aspects of emotional dysregulation at such times argue more for a focus on the nature of accompanying (and more differentiating) depressive episodes. Those with a bipolar disorder are most likely to experience psychotic or melancholic depressive episodes, whereas those with BPD experience non-melancholic depressive episodes, generally triggered by environmental stressors rather than being autonomous.

Bassett\(^7\) nominated other differentiating factors in those with BPD and bipolar disorder: respective high and low rates of early sexual abuse; limited v. more extensive loss of white and grey matter; normal v. reduced activity of cuneus and lingual gyri; absence v. presence of mitochondrial dysfunction; and non-response v. response to a mood stabiliser. In his extensive review of what he summarised as ‘separate disorders’, Bassett argued that, as those with bipolar disorder do not have a disrupted sense of self or an incapacity to maintain satisfying interpersonal relationships, and as core features are mood weighted, it is intrinsically a mood disorder. By contrast, he argued that the core elements of BPD
are an ‘emotionally noxious sense of the self’ – manifested by recurring fears of abandonment and chronic emptiness – therefore positioning such conditions at the level of disordered personality functioning.

Borderline personality disorder and bipolar conditions appear, then, to have sufficient distinctive characteristics to argue for their nosological distinction. Imprecision in their clinical differentiation presumably reflects either diagnostic difficulty or true comorbidity (quantified by Paris et al in the order of 10%).

**Is BPD a unipolar mood condition?**

The long-standing binary classificatory model positioned contrasting endogenous/psychotic/melancholic v. neurotic/reactive/non-melancholic depressive conditions. Ascriptions for the first subset include a strong genetic contribution, biological underpinnings, no distinctive personality predisposition, and a set of overrepresented features such as a non-reactive and anhedonic mood, diurnal mood and energy variation and psychomotor disturbance (together with the presence of delusions and/or hallucinations in the psychotic subset) – a differing profile to depressive episodes characterising BPD.

The contrasting non-melancholic depressive disorders are principally viewed more as a consequence of antecedent stressors and/or a vulnerable personality style, and might therefore be positioned as primary depressive syndromes (Axis I), personality-based (Axis II) or straddling both axes, and so allowing, in effect, a ‘spectrum model’. In detailing such a spectrum model, Manicavasagar and I8 positioned neurobiological processes as shaping personality style and, when the individual is stressed or depressed, such personality nuances being amplified to shape the clinical depressive phenotype. For example, individuals with high-trait ‘internalising’ anxiety manifest an anxious disturbance (together with the presence of delusions and/or hallucinations in the psychotic subset) – a differing profile to depressive episodes characterising BPD.

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Recent DSM manuals (including DSM-5) have only one such ‘spectrum’ condition – ‘atypical depression’. In an earlier paper,7 my colleagues and I charted its history from initially being positioned as a primary anxiety condition (Axis I), personality-based (Axis II) or straddling both axes, and so allowing, in effect, a ‘spectrum model’. In detailing such a spectrum model, Manicavasagar and I8 positioned neurobiological processes as shaping personality style and, when the individual is stressed or depressed, such personality nuances being amplified to shape the clinical depressive phenotype. For example, individuals with high-trait ‘internalising’ anxiety manifest an anxious disturbance (together with the presence of delusions and/or hallucinations in the psychotic subset) – a differing profile to depressive episodes characterising BPD.

This paper seeks to argue against any positioning of BPD as a mood disorder by weighting phenomenological and other differences. Difficulties in clinically differentiating BPD and bipolar disorder (especially bipolar II disorder) can be reduced by weighting a number of longitudinal and cross-sectional differences between the two conditions. In conceding that some patients may possess both conditions this does not alone argue for their merging in one diagnostic class.

**Conclusions**

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**References**

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